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APPENDICITIS AS A SEQUELA OF TONSILLITIS.

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The occurrence of appendicitis as a sequela of tonsillitis has received less attention from clinicians in America and Great Britain than its practical importance warrants. Most of the standard works on medicine and surgery make no reference to it, and it is seldom noticed in current medical literature. This is the more remarkable, since the importance of the tonsil as the port of entry of many forms of systemic infection, and as the starting-point of disease in distant organs, has been well established in recent years in numerous contributions by recognized authorities. In this connection one need only enumerate general sepsis, acute rheumatism, chorea, endocarditis, pneumonia, pleurisy, empyema, meningitis, nephritis, urethritis, orchitis, thyroiditis, and cholecystitis, all of which have been definitely traced to acute tonsillar infections.

About a year ago a patient entered my service in St. Michael's Hospital, a young woman, who developed an abscess in the upper lobe of the right lung a week after an operation for removal of the tonsils during the subsidence of an acute angina.

The relationship between acute rheumatism and appendicitis has

frequently been noted, and when one recalls how often tonsillitis precedes the former, the etiological connection of tonsillitis with appendicitis is more readily apparent. To Sir James Grant, of Ottawa, is due the credit of reporting in 1893, the first modern instance of the association of appendicitis and rheumatism.[1]

The same year Kelynack directed attention to the occurrence of appendicitis secondary to tonsillitis.[2]

In Europe the association of the two diseases has been more systematically studied from both the experimental and clinical stand-points, and consequently more generally recognized by the medical profession as a whole. The similarity in structure between the tonsil and appendix has frequently been pointed out and given as an explanation of their liability to similar infections. While the general trend of opinion among those who have studied the question is to recognize the etiological association of the two diseases, there is considerable diversity of view as to its frequency and the route of transmission of the infection. The experimental and clinical investigation of the latter has elicited much information bearing upon the pathology of appendicitis, especially as to whether the disease is due to a local infection from the bowel or to a hematogenous infection, with secondary localization in the lymphoid tissue of the appendix.

Adrian, in 1901,[3] succeeded in producing appendicitis in rabbits by the intravenous injection of staphylococci, streptococci, pneumococci, B. typhosus, B. coli, B. anthracis, and the tubercle bacillus. He regards the appendix as a point of election for the localization of general infections.

Subsequently, Tedesco[4] investigated experimentally the significance of tonsillar infection, using streptococci, staphylococci, and B. anthracis. He concluded that pyogenic germs from the pharyngeal ring can in rabbits lead to embolic processes in the parenchyma of the appendix, and that it is therefore possible for hematogenous infection to produce characteristic follicular disease of the appendix.

Mori[5] concludes that appendicitis may originate by way of the blood channels.

Kretz[6] believes that almost every case of appendicitis is in causal connection with angina through hematogenous infection.

The conclusions of these authorities have been called in question

[1] New York Med. Record, 1893, I, 609.

[2] A Contribution to the Pathology of the Vermiform Appendix, London, H. K. Lewis, 1893.

[3] Mitteilungen aus der Grenzgebiete d. Med. u. Chir., Band vii, S. 407.

[4] Experimentelle Beiträge zur die Aetiologie der Epityphlitis, Deutsch. Zeitschr. f. Chir., 1904, Band lxx.

[5] Eine experimentelle Arbeit über die Aetologie d. Epityphlitis, Deutsch. Zeitschr. f. Chir., 1904, Band lxx.

[6] Ueber die Aetiologie der Appendicitis, Verhandl. der deutschen Pathologischen Gesells., 1910.

by Ghon and Namha[7] as the result of their experiments, as well as by Aschoff,[8] Oguro,[9] and others.

Other more recent investigations and clinical reports, however, recognize the possibility of hematogenous infection and the importance of tonsillitis in relation to appendicitis.

Schroetter[10] thinks that an etiological connection between angina and appendicitis has been established, and that this is confirmed by the prevalence of appendicitis a few weeks after the changeable seasons, when angina and throat troubles are especially common.

Kelly[11] recognizes the relationship of appendicitis to tonsillitis, and states that in the surgical clinic of the Johns Hopkins Hospital there were 3 instances of its occurrence out of 91 cases of simple acute appendicitis.

Very convincing evidence in favor of the hematogenous origin of appendicitis has been brought forward by Poynton and Paine,[12] who believe that they have furnished almost conclusive proof that appendicitis may result from streptococcal invasion through the blood stream from a follicular tonsillitis.

Oguro thinks there is no doubt that hematogenous infection does occur in some cases, but less frequently than Kretz maintained. Boit and Heyde[13] express a similar opinion.

Heile[14] does not deny the possibility of hematogenous infection, but it seems to him more natural and simple to assume that the infective agent reaches the appendix and peritoneum from the intestines.

Haeberlin[15] thinks there is no doubt that hematogenous infection occurs after angina and scarlet fever, but that the number of cases in which it plays a role is very limited.

Heyde[16] reports a case of appendicitis following angina, and while he believes a hematogenous origin is uncommon, yet the importance of angina as a predisposing factor should be recognized.

Rèthi[17] says that recently it has been recognized that the tonsils comparatively often form the starting-point of general infections,

[7] Zur Frage über die Genese der Appendicitis, Beitr. z. Path. Anat., 1911, Band lii, S. 120.

[8] Die Wurmfortsatzentzundungen, Jena, 1908.

[9] Ueber die Aetiologie u. Pathogenesis der Epityphlitis, Virchows Arch., 1909, B and cxcvii, 548.

[10] British Med. Jour., 1907, i, 534.

[11] Appendicitis and Diseases of the Vermiform Appendix, 1909, p. 150.

[12] Further Contribution to the Study of Rheumatism, Lancet, 1911, ii, 1189; also The Etiology of Appendicitis as a Result of Infection, Lancet, 1912, ii, 439.

[13] Experimentelle Untersuchungen zur Aetologie der Wurmfortsatzentzundungen, Beitr. z. klin. Chir., 1912, lxxi, 271.

[14] Zur Pathogenesis des Appendicitis, Arch. f. klin. Chir., 1909, Band xc, 190.

[15] Zur Entstehung der Wurmfortsatzentzundungen, Deutsch. med. Wchnsch., 1909, xxxix, 394.

[16] Untersuchungen zur Aetiologie der Wurmfortsatzentzundungen; Beitr. z. klin. Chir., 1911, Band lxxii, 1.

[17] Die Tonsillen als Ausgangstelle der allgemeine Sepsis, Wiener med. Wchnsch., 1912, lxii, 446.

including appendicitis. An instructive case is mentioned by Deaver of an appendicular abscess following an attack of diphtheria. L. Herdelet[18] reports two cases of appendicitis following tonsillitis. Many other cases might be referred to, but sufficient data have been brought forward to indicate the general trend of opinion, and the wide recognition of the association of tonsillar infections with appendicitis which has occurred in recent years.

Tedesco[19] says he has produced embolism and necrosis of the appendicular follicles experimentally in rabbits. He further states that if appendicitis is immediately preceded by tonsillitis, rapid necrosis of a follicle in the appendix, with perforation or gangrene, is to be anticipated, and therefore operation should be performed early.

Tedesco and also others have referred to the tendency which these cases following tonsillar infection have to develop extremely acute symptoms.

He says that when a staphylococcic infection becomes generalized it is likely to prove fatal. This, however, is by no means an invariable result.

It would therefore appear to be established on experimental as well as clinical data that a hematogenous origin of appendicitis does occur, a fact of much importance in explaining the unusual course at times taken by the latter disease. The clinical relationship of appendicitis and tonsillitis, however, does not depend for its acceptance upon the theory of the hematogenous origin of the appendicular infection. It has been shown that appendicitis can be produced in rabbits by feeding pathogenic bacteria (Chastenet),[20] so that the possibility of infection from the tonsil by way of the alimentary tract is readily apparent.

Some seven years ago my attention was first directed to the occurrence of appendicitis as a sequela of tonsillitis by a case which came under my observation, the history of which is briefly as follows:

H. T., aged nineteen years, student. Father and mother both living and well, aged seventy-five years. They had ten children, all living and well. One brother and one sister had suffered from stomach trouble, with much distention at times, the brother having had a gastro-enterostomy performed by Dr. Stiles, of Edinburgh. The patient, though not robust, had always had good health.

On January 10, 1907, he felt chilly, "out of sorts," had slight headache, general pains, anorexia, and sore throat. When I saw him the next day, examination showed that the tonsils were enlarged and that there was follicular inflammation. The pharynx, fauces,

[18] Gaz. Hebd. Soc. Méd. de Bordeaux, April 12, 1914 (British Med. Jour., June 6, 1914).

[19] Wiener med. Wchnsch., January 9, 1914, p. 82.

[20] Thèse de Paris, 1897.

soft palate, and uvula were also swollen, dusky red in color, and there was a slight sticky exudate on the pharynx. Temperature, 102°; pulse, 100.

Rest in bed, gargles, and aspirin were prescribed. A culture taken from the throat showed Staphylococcus pyogenes aureus.

On the following day the condition of the throat was considerably better; the temperature was 102.5° and the general condition remained much the same. On January 13, I was called to see him at 4 A.M., owing to the development of severe epigastric pain, gastric distention, and belching of gas. There was some tenderness over the epigastric and left hypochondriac regions, and the stomach was so distended that the outline of the great curvature was plainly visible, moving up and down with respiration. The bowels had been moving freely. Temperature, 100°; pulse, 84. There was absolutely no pain, tenderness, distention, or rigidity over other areas of the abdomen. The throat condition had greatly improved. The pain disappeared in a short time, and for two days the patient was apparently much better; the throat had cleared up. Evening temperature, 98.1°; pulse, 80.

On the evening of January 15, there was a recurrence of pain, and he was sent to the hospital on January 16. On admission, at 10 A.M., the temperature was 98°; pulse, 84. Gastric distention, with retching, but inability to vomit, had recurred, and the patient looked ill, out of proportion to his other symptoms.

Dr. G. A. Bingham saw him in consultation at 4 P.M. The temperature was 97.3°; pulse, 84; respirations, 18. There was distention in the epigastric region, but no pain, tenderness, rigidity, or distention elsewhere. He considered operation unwarranted. The throat was practically well.

During the night general abdominal pain, tenderness, and distention developed. The temperature increased to 99.2°; the pulse to 140. The condition unfortunately was not reported to me until my arrival at the hospital in the morning. The patient at that time had all the signs of a generalized peritonitis. An operation was performed and a gangrenous appendix found, with a generalized peritoneal infection. The patient died the same evening. No autopsy was obtained.

Urinalysis on the morning of operation was negative; leukocytosis, 30,000.

The case was so unusual in its course, so distressing in its outcome, and presented so many puzzling features, that I have since followed the subject with much interest.

Dr. Lorne Main, of Dundas, has kindly supplied me with notes of another case, as follows:

J. W., aged twenty-two years. The patient was a robust young man, weighing 190 pounds. He had had an attack of gonorrhea three years previously and a syphilitic infection one year before the

illness to be discussed. On March 4, 1911, he sought treatment for acute follicular tonsillitis. His temperature was 103.5°; pulse, 96. He complained of slight pain and tenderness in the right iliac region, which had, however, disappeared by the next day. Under the usual treatment his temperature became normal on March 8, although he was not allowed out until March 14. He felt very well, and went out for a walk. The tonsillitis had entirely subsided. About 9.30 in the evening he complained of headache and feeling tired; the temperature was normal; pulse, 80. On the next day, March 15, he was about the house; pulse and temperature both normal. The bowels had not moved for two days. At 3 A.M. on March 16, he was attacked by severe abdominal pain.

Dr. S. Cummings operated at 3.30 P.M. There was a gangrenous appendix, with what appeared to be gangrenous areas in the adjacent portions of the small and large bowel. The patient died at 1.30 A.M., March 17.

Other observers have reported cases in which the tonsillitis preceded the appendicitis by only a few hours, and it has been noted that rheumatism may develop coincidently with or in some cases follow the onset of appendicitis.

In both these cases a remarkable feature was the lateney of symptoms referable to the appendix. The absence of fever or increase of pulse rate until the sudden development of fulminating symptoms may also be noted. In both cases a gangrenous appendix was found within about twelve hours after the onset of symptoms which might be definitely referred to the appendix. In my case the early pain and tenderness were both referred to the epigastric and left hypochrondriae regions, especially the latter, and the stomach showed extreme distention. Dr. Bingham and I both attributed the epigastric pain and tenderness to the extreme distention of the stomach.

Neither Dr. Bingham nor myself could detect either tenderness or rigidity over the region of the appendix a few hours before the symptoms of general peritonitis developed.

In appendicitis my own experience is that whereas pain may be referred to the epigastrium or other areas, yet tenderness on pressure and at least some rigidity over the region of the appendix, are almost invariably to be demonstrated by careful examination.

Unfortunately a postmortem examination was not permitted, but I[21] was of opinion that there was a generalized infection, with involvement of the lymphoid tissue, in both the stomach and appendix to account for the symptoms.

[21] The above opinion was expressed seven years ago. Since this paper was read in July, 1914, Edward C. Rosenow has published his article on the Bacteriology of Appendicitis and its Production by Intravenous Injection of Streptococci and Colon Bacilli, Journal of Infectious Diseases, March, 1915, xvi, 240, in which he proves, experimentally, that such multiple hematogenous infection of the appendix, duodenum, small intestine, and stomach may occur.

Another possible explanation of the early gastric symptoms is that they were due to a generalization of the infection from the tonsil with secondary involvement of the appendix, or to an infection of the appendix by way of the alimentary tract; in either case the acute early process subsiding with coincident improvement for some days of the pulse, temperature, gastric condition, and general symptoms. After smouldering in the appendix the infection suddenly became active with the onset of fulminating symptoms.

In Dr. Main's case there was definite evidence of involvement of areas in the small and large intestine, as well as of the appendix, suggestive of a hematogenous infection. The evolution of symptoms would suggest that an infection from the tonsils of the lymphoid tissue in other areas of the alimentary tract had occurred, and after smouldering for a time suddenly became active with the onset of fulminating symptoms.

In this case it is interesting to note that the patient complained of slight pain and tenderness in the right iliac region, which, however, had disappeared by the next day (March 5). The acute fulminating symptoms appeared March 16 after the patient was apparently well, suggesting that in the interval the infection in the appendix had remained dormant.

Dr. Arthur Wright has furnished me with notes of the following case from Dr. G. A. Bingham's clinic in the Toronto General Hospital.

Mrs. W., aged forty-seven years, came under observation on April 1, 1914. She had for the previous three days been suffering from pain in the right lower abdomen, radiating to the back. She had previously suffered from acute rheumatism (more than thirty years ago), and two years previously had been treated in the Toronto General Hospital for cardiac trouble. There was profuse uterine discharge, in which gonococci could be demonstrated, and there was also retroversion of the uterus. A parovarian cyst was removed.

On June 10 the patient complained of a sore throat, and on June 12 the tonsils were found to be enlarged and reddened, and there was marked pain on swallowing. On June 16 there was abdominal pain of sudden onset, commencing in the umbilical region, and followed by vomiting. The pain gradually became localized over the region of the appendix, but the temperature and pulse remained normal.

On June 17 the symptoms had become more severe, and pain, tenderness, and rigidity of the right rectus were present to a marked degree, and there was a mass in the appendicular region. Evening temperature 103°; pulse, 114. Leukocytes 21,400. The tonsils were still inflamed.

An operation was performed at 8.30 P.M. and a gangrenous appendix, in a retrocecal position, was removed. Recovery occurred, and on June 26 the patient no longer complained of pain or soreness of the throat.

In conclusion, I= would like to emphasize the following points:

1. The importance of bearing in mind the liability of appendicitis to follow acute tonsillitis.

2. That the appendicular involvement may be only part of a generalized infection, hence the gravity of such cases out of proportion to the local symptoms.

3. The tendency for such cases to be atypical in their clinical course, and after smouldering, to suddenly develop fulminating symptoms.

4. Chronic tonsillar infections should be kept in view as the possible cause of similar infections of the appendix.

5. That whereas at least some degree of local tenderness and rigidity are almost always to be elicited on careful examination of the abdomen in the right iliac region in acute appendicitis, in rare cases these signs may be absent.

LIGATION OF THE PORTAL VEIN IN SUPPURATIVE PORTAL PHLEBITIS.

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The problem of attacking pylephlebitis along the lines employed in septicemia of otitic and of uterine origin, has as yet attracted no great attention.[1] A. G. Gerster,[2] commenting on this disease, says: "The evacuation of septic thrombi from the jugular vein in mastoid disease has yielded such excellent results that the application of the principle to the portal vein would be natural and logical. But the anatomical relations, while very favorable in the former instance, are just the reverse in the latter. Only a short piece of the portal vein, that situated in the hepatoduodenal ligament, is approachable. The two mesenteric veins and the splenic are practically inaccessible. Hence, though phlebotomy of the portal trunk in the hepatoduodenal ligament is not impossible, the evacuation of thrombi by flushing through a catheter seems to be too problematic, not to mention the technical difficulties the surgeon might encounter in the closure of the phlebotomy wound."

= Read before the Canadian Medical Association, St. Johns, July, 1914.

[1] H. Kehr, Chirurgie d. Gallenwege, 1913. Suppurative thrombi in the portal vein have never been removed operatively. Such a procedure might possibly have a beneficial effect.

[2] Trans. Am. Surg. Assoc., 1903.